PERIPHERAL AUDITORY ASSESSMENT IN

MINOR HEAD INJURY

A PROSPECTIVE STUDY IN TERTIARY HOSPITAL

A DISSERTATION SUBMITTED IN PART FULFILLMENT OF

THE RULES AND REGULATIONS FOR THE M.S (BRANCH-IV)

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CERTIFICATE

This is to certify that the dissertation PERIPHERAL AUDITORY ASSESSMENT IN MINOR HEAD INJURY is the bonafide work of Dr. Lingamdenne Paul Emerson carried out in the DEPARTMENT OF ENT at CHRISTIAN MEDICAL COLLEGE VELLORE, TAMILNADU, under the guidance of Dr.John Mathew, Professor and Ag Head, DEPARTMENT OF ENT UNIT-II, for the M.S (Branch -IV) OTORHINOLARYNGOLOGY EXAMINATION OF THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY, to be held in March-2008.

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CONTENTS

INTRODUCTION

Hearing loss is a common problem encountered in ENT practice. Recognition of hearing loss as a problem by the patient usually occurs when speech frequencies are affected or when there is a sudden hearing loss. Hearing loss following head trauma or head injury is a major medical problem in adults[1] as well as children[2], which may go unnoticed when the hearing loss does not affect speech frequencies. Sensorineural hearing loss at high frequencies is a frequent finding associated with head injuries[3]. Hearing impairment can be due to central or peripheral causes, middle ear or cochlea being the most common site of peripheral injury. The most pronounced injury is fracture of temporal bone[4]. In both clinical and animal experimental studies it has been shown that there are various sites of pathology ranging from hair cell damage and degeneration of the organ of corti,[5] ischemia of the 8th nerve to damage of central auditory pathways (due to compromise of blood supply to the inner ear) either partly or totally[6]. In most cases hearing impairment dissipates during subsequent post traumatic period, but some times it may persist or progress.

The causes of progression of hearing loss are not well known. Several explanations have been proposed such as development of perilymphatic fistula[7,8] secondary degenerative changes in cochleaError: Reference source not found following inner ear concussion possibly due to consequences of pre-existing autoimmune disease[9] (or trauma itself may initiate such a reaction towards specific inner ear proteins).

Progression of hearing loss can be attributed to the synergistic effects between trauma, noise exposure[10], medication[11] and meningitis.

Minor head injuries[12] constitute a major portion of all accidents. Evaluation of these patients revealed presence of hearing loss in the high frequency range. Thus auditory assessment is needed in this group of patients.

In India roads are used not only by modern cars and buses, along with locally developed vehicles for public transport (three-wheeled scooter taxis, auto rickshaw's), scooters and motorcycles, bicycles, but also by rickshaws, and animal or human drawn carts that has resulted in disproportionate increase in road traffic accidents in our country compared to developed countries.

Evaluation of hearing loss in patients who sustain minor head injury has not been done in the Indian subcontinent. Therefore, this prospective study was done to evaluate the incidence of hearing loss and estimate its progression or regression by serial audiometric assessment over a period of six months.

AIMS AND OBJECTIVES

1. To evaluate the prevalence and types of hearing loss in patients who sustained minor head injury.

2. To define its natural history and to estimate the progression/regression of hearing loss by serial assessment of hearing.

3. To evaluate the presence or absence of Distortion product otoacoustic emissions

REVIEW OF LITERATURE

Hearing loss is a well known entity following head injury. The severity of hearing loss may vary depending on the severity of the head injury. The severity of injury is measured clinically using the Glasgow coma scale scoring system [13](G.C.S).

Glasgow coma scale scoring system Error: Reference source not found

The Glasgow Coma Scale was first published by Teasdale and Jennett in 1974. Several years later that was modified by Jennett and Teasdale[14] and by Rimel et al[15,16] This scoring system provides the best measure of severity of head injury. The score is the sum of the scale's three measures of eye opening, best motor and verbal responses. This ranges from a score of 3 for a patient with no motor, verbal response or eye opening to painful stimuli, to 15 for a patient who is oriented, follows commands, and has spontaneous eye opening. Patients, who do not follow commands, speak or open their eyes, with a score of 8 or less, are by definition in coma. Head injury is defined as mild when the GCS score is either 13 or 14–15, moderate by a score of 9–12 or 13, and severe by a score of 3–8. The GCS score on admission, and its prognostic usefulness, are easily confounded by other factors particularly substance misuse, but sequential monitoring after admission plays a crucial role in detecting early deterioration and in its management.

Minor head injuries were therefore defined as a GCS score of 13–15 in this system. Culotta[17] in 1996 following a retrospective study found that patients with a GCS score of 13–15, represented a heterogeneous group of patients with statistically significant different head tomography abnormalities. Culotta on the basis of their findings suggested separating patients with GCS score 13–14 into a different category from patients with a GCS score of 15, thus effectively redefining minor head injury. These findings were confirmed by a similar study by Gomez et al[18]. Hsiang et al[19] in1997, on the basis of a cohort study of 1360 patients with GCS score of 13–15 suggested that this group of patients could also be divided into two subgroups, mild head injury and high risk mild head injury. Mild head injury is defined as GCS 15 without radiographic abnormalities, high risk mild head injury being defined as GCS 13–14, or a GCS 15 with acute radiographic abnormalities. More recently Swann and Teasdale[20] recognizing the limitation of the GCS with regard to minor head injury have suggested another sub classification. Mild head injury is defined as GCS 13–14. Minor head injury is defined as GCS score of 15. The authors recognized in their monograph that this is a somewhat arbitrary definition. However in Clinical practice GCS is used in evaluation of Head injury.

Glasgow coma scale and scoreError: Reference source not found, Error: Reference source not found :

Clinical parameter

EYE OPENING	BEST MOTOR RESPONSE	VERBAL RESPONSE
Spontaneous - 4	Obeys commands - 6	Oriented - 5
To speech - 3	Localizes to pain - 5	Confused, Disoriented-4
To pain - 2	Withdraws (Flexion) - 4	Inappropriate words - 3
None - 1	Abnormal flexion -3	Incomprehensible sounds -2
	Abnormal extension - 2	No verbal response -1
	No motor response - 1	

Glasgow coma score: (E+M+V) = 3–15

INCIDENCE OF ROAD TRAFFIC ACCIDENTS IN INDIA

In India, in the year 2000[21], official statistics revealed that 80,118 persons died and 3,42,200 were injured in road traffic accidents. However, this is an underestimate, as not all accidents are reported to the police.

A study done in Haryana (India) recorded all traffic-related injuries and deaths through bi-weekly home visits to all households in 9 villages for a year[22]. This study showed that the ratio between critical, serious and minor injuries was 1:29:69.

The state of TamilNadu[23] ranks second among States with the highest number of road accidents, according to the national statistics. The National Highways, which run across 4,500 km in Tamil Nadu, accounts for 35 percent of fatal accidents and the State highways for 32 percent. While last year saw an average of 153 accidents every day, this year, the number has increased to 165 a day.

The North Arcot District Health Information news letter [NADHI][24] March 2006 to April 2007, revealed that in Vellore District majority of road traffic accidents occur on Village roads and state highways with majority of injuries sustained by people on two and three wheeler vehicles.

According to NADHI there are on an average two fatal accidents occurring per day with more than three minor accidents reporting to major hospitals in Vellore district alone.

Data for alcohol abuse and other drug involvement in road traffic accidents are not available for all Indian cities. Studies done in Delhi[25], Bangalore[26] documented that about one third of two-wheeler riders admitted to a neuro-surgery ward were alleged to be under the influence of alcohol at the time of accident. It is clear that much more work needs to be done to understand the exact role of alcohol and other drugs in road traffic accidents in India. However, the limited information available suggests that this may be a significant risk factor.

Mild traumatic brain injury[27] is one of the most common neurological disorders with only migraine and herpes zoster having higher incidences and only migraine having higher prevalence. The severity of traumatic brain injury must be defined by the acute injury characteristics and not by the severity of symptoms at random points after trauma.

Mild traumatic brain injury is characterized by the following

1. Head trauma may be due to contact forces or due to acceleration/deceleration trauma.

2. The duration of unconsciousness is brief usually seconds to minutes and in some cases there is no loss of consciousness but simply a brief period of dazed consciousness

3. When the patient is evaluated in the Emergency room or at the scene the GCS must be 13-15 by common definition.

4. Whether a patient is briefly unconscious or not, confusion with amnesia is present by definition for less than 24 hours, but usually for minutes to a few hours.

Patients with skull fracture on radiography[28] were at a greater risk of having a hematoma and further deterioration.

Definition of a mild head injury[29]

A mild head injury can be defined as an injury caused by blunt trauma and/or sudden acceleration /deceleration which produces a period of unconsciousness for 20 minutes or less, a Glasgow coma scale scoreError: Reference source not found of 13-15, no focal neurological deficit, no intracranial complications and computed tomographic findings limited to a skull fracture without evidence of contusion or hematoma.

Despite the dissemination of information that is available on diagnostic criteria, controversies still exist in defining mild head injury and collecting patients.

Estimating the duration of unconsciousness is difficult when witnesses are not available, second if the patient is intoxicated at the time of hospital admission it can obscure the assessment of severity of head injury.

Hearing Loss in Head Injury

Deafness due to head injury is known since ancient times, the earliest account of which is the Edwin Smith papyrus[30], the world's earliest known medical document, written around 1600 BC, but thought to be based on material from as early as 3000 BC. It is a textbook on trauma surgery, and describes anatomical observations physical examination, diagnosis, treatment, and prognosis of numerous injuries in exquisite detail. The symptoms and signs of head injury were given in considerable detail. It was noted that brain injuries were associated with changes in the function of other parts of the body. Feeble pulse and fever are associated with hopeless injuries and deafness as well as aphasia are recognized in fractures of the temporal region.

Sushrutha[31] who is known as "Father of Surgery" in India, even though he does not attach significance to the brain, however, considers head as the centre of all special senses and describes certain cranial nerves connected with specific sensory functions. He described two nerves lower down the back of the ear (vidhura), which, if cut, produce deafness; a pair of nerves (phana) situated inside the two nostrils, which if cut, cause anosmia .

A pair of nerves below the outer end of the eye-brow, near the external corner of the eyeball (apanga) which if cut, cause total blindness.

The incidence of hearing loss in head injury was reported by Alexander and Scholl[32] in 1938 as 31% of which 80% of patients had skull fractures and 10 % had brain concussion.

In 1939 Grove[33] reported an incidence of 32.6% of sensorineural hearing loss after head injury. The incidence according to Muller[34] is 13% and according to Barber[35] 24%.

Gurdijan[36] reported hearing loss in 129 patients of a total of 718.

In 1975 Fradis and Podoshin[37] found hearing loss in 22.2% of patients.

Griffith[38] in 1979 reported an incidence of 56% of sensorineural deafness in cases of mild head injury.

George G Browing[39] in 1982 assessed patients with minor head injury with no evidence of temporal bone fracture. According to him there is no difference in thresholds in pure tone audiometry between study group and controls. His controls were men and women admitted with other injuries but no history or clinical signs of head injury.

According to GriffithError: Reference source not found there may be difference in outcome depending on the type of violence. A blow to the head with a soft object seems to cause less damage to the hearing system than a blow to the head with a

hard object with the same power at impact. The greater hearing loss according to him is due to an acoustic component.

The site of injury is important, frontal injuries resulting in a comparatively low incidence of hearing loss as compared to temporal blows. The type of audiogram recorded is an important indicator in assessing prognosis. Vertigo according to them has its own separate etiology and should be assessed and treated separately and not as a part of the post concussion syndrome.

The incidence of hearing loss and vertigo in the immediate post injury period is very high 56% and 24% with 8% in controls with recovery within three months. Accordingly the mechanism of low frequency hearing loss is seen in patients with hydrops and suggests a similar peripheral mechanism. He stated that the lesions lie in the peripheral labyrinth due to edema or hydrops both of which subside with excellent prognosis. High frequency hearing loss may be caused by concussion and intense acoustic stimulation, concussion being reversible.

M R Abd al Hady[40] in 1990 reported a 20% hearing loss in 30 patients with minor head injury.

Vartianenn[41] described a series of 61 children with blunt head injury in which 30% had hearing loss.

William D Zimmerman[42] reported a hearing loss of 48% in children with head injury.

E B Dorman[43] noted that immediately after trauma 25% had sensorineural hearing loss.

The site of hearing impairment can be peripheral or central although the peripheral structures i.e., the middle ear and cochlea represent the most common site of injury.

Nassulphis[44] found damage in the Reissner membrane and degeneration of the organ of corti in the spiral ganglion and cochlear nerve in several patients suffering from hearing loss following head injury.

According to Schuknecht and Davison[45] auditory symptoms following head injury can be grouped according to the classification of labyrinthine damages which are:

1. Longitudinal fracture of temporal bone

2. Transverse fracture of temporal bone

3. Labyrinthine concussion

Labyrinthine concussion may be described as perceptive deafness and vertigo resulting from a blow to the head without fracture of bony labyrinth capsule.

The nystagmus is positional and may persist for several months. The underlying pathology was thought to be due to injury to the utricle and saccule.

Histopathological evidence showed rupture of the membranous walls of the utricle and saccule and degenerative changes in the macula of the saccule. This injury is commonest in ear with longitudinal fracture of the temporal bone, secondly in a ear opposite a temporal bone fracture and thirdly in a head injury with no evidence of skull fracture. According to them to produce labyrinthine concussion a head injury must be severe enough to cause loss of consciousness.

Hearing loss is worst in the high frequency range and the peak loss is usually at 4000 Hz. The vertigo attacks are of postural type as described for patients having longitudinal fracture.

The animal experiments show that the deafness is due to injury of the organ of corti, identical with that which results from a shock pulse in the air as a bomb blast or a pistol shot. They found that it results in violent displacement of the basilar membrane and organ of corti, that both reversible and irreversible cellular injuries result. They estimated hearing loss in animals, cats subjected to head injury.

The hearing loss was estimated by audiogram and compared with a cochlear chart and found that the primary effect of trauma is to the organ of corti and the nerve degeneration is secondary.

It is in fact the presence of damage to the organ of corti which ruled out nerve injury as a primary effect.

The slightest detectable histological changes consisted of anatomical derangement of outer hair cells and their supporting cells.

In mild injuries the outer hair cells which are normally tall and rectangular appeared shorter and wider and the nuclei were smaller and the chromatin was condensed.

In severe lesions there was a loss of external hair cells and the beginning of cytological changes in the Dieters cells and the supporting cells, further progressive stages of injury consisted of flattening of the organ of corti and finally its complete disappearance.

In labyrinthine concussion histological examination of the auditory system revealed the significant pathological changes to lie in the cochlea, when there was damage to the organ of corti severest in the upper basal turn, the region serving 4000Hz frequency.

Thus according to them partial permanent deafness occurs in about 50% of patients who sustain a blow to the head to produce unconsciousness.

Even a mild head blow without loss of consciousness can occasionally result in deafness.

According to Makashima and Snow[46], experimental findings by means of assessment of preyer reflex and cochlear potentials in guinea pigs after stimulating head injury by shaking them in a padded cage till they became unconscious showed that in animals which did not have fracture of the skull showed hemorrhage in and laceration of the 8th nerve where it exits from the medulla oblongata.

Animals killed after 6 days and 30 days showed slight to moderate degeneration of outer hair cells and Hensens cells in the apical and middle turns of the cochlea, changes in the Stria vascularis were minimal.

The oto-neurological manifestations vary from patient to patient with head injury. Variability exists in type, severity and mode of onset of symptoms and signs. In some patients deterioration of hearing and vestibular functions occurs immediately after head injury and it may be transient or permanent. In other patients the symptoms may not manifest until later and deterioration of function may continue. According to them these facts suggest that there are various forms of trauma in the temporal bone and central pathways could be responsible for the deterioration of function.

In a study done by E B Dorman et alError: Reference source not found the hearing loss was noted to be due to cochlear dysfunction. No 8th nerve or central abnormalities were detected. Various hypotheses have been put forward to explain the hearing loss that appears after brain concussion.

CAUSES OF HEARING LOSS

GrooveError: Reference source not found felt that bleeding in the inner ear was the cause whereas Uffenorde[47] stated that stretching of the fibers of the cochlear nerve in the internal auditory canal bought on the hearing loss.

On the other hand, Schuknecht and Woellner[48] found that it is possible to destroy 75% of the cochlear nerve fibers that are innervating an area of the cochlea, without causing any change in the hearing threshold of that area.

According to Andrew T Lyos et al7 in case of temporal bone fracture, immediate profound hearing loss may be caused by avulsion of the nerve or severe damage to the membranous labyrinth. Concussion directly to the otic capsule or acoustic trauma via the ossicular chain is well described. If it is not severe, it produces transient cochlear hyperemia resulting in temporary threshold shift.

Feldman[49] stated that, sudden hearing loss with delayed onset can also occur following head trauma, thus it may be due to the sequelae consisting of perilymph fistula in one of the windows or a fracture of the labyrinthine capsule, which may manifest for the first time after a period of years or even decades.

Allison M Scott [50] found that in addition to low and high frequency hearing loss audiograms with single and double sensorineural notches in mid frequency region may be related to head trauma.

According to Per-Olof Bergemalm[51] in cases of closed head injury 74% of patients showed progression of > 15dB HL which was significantly greater than the spontaneous progression in the control group. Age and temporal bone fractures were risk factors for progression but not brain contusion only or Swedish Reaction Level Scale (RLS)[52]. They found an association between early PTA (Pure tone Audiometry) and progression as well as regression i.e. poorer the initial PTA the greater the progression or regression indicating the increased instability of the auditory system.

The cause of progression is usually unknown. One of the possibilities is the development of perilymphatic fistula, other reasons may be secondary degenerative changes in the cochlea following inner ear concussion and hypoxia following disturbance of micro-circulation.

It has also been speculated whether progression is due to pre-existing autoimmune disease or whether the trauma itself may initiate such a reaction towards specific inner ear proteins.

There may also be synergistic interaction between trauma and the effects of noise exposure[53] and the use of oto-toxic agents and medicationError: Reference source not found.

Ludwig podoshinError: Reference source not found found that in cases of head injury with brain concussion without skull fracture 14.7% suffered from hearing loss of which 12.7% was sensorineural and 87% was conductive type.

In cases of fracture of skull with out fracture of temporal bone damage was most marked in frequencies of 4000 Hz.

According to Griffith Error: Reference source not found10% of the total hearing loss is of conductive type and 90% is of sensorineural type .

E.B.DormanError: Reference source not found reported a 25 % sensorineural hearing loss and in no case it was greater than 30 decibels.

VartiannennError: Reference source not found reported 16.3% conductive hearing loss and 13% had sensorineural hearing loss in cases of head injury in children.

According to ZimmermanError: Reference source not found 32% had conductive hearing loss and 16% of patients had high frequency sensorineural hearing loss.

Ludwig podoshinError: Reference source not found reported that conductive hearing loss due to head injury usually disappears in two months, if it persists ossicular discontinuity must be suspected. Improvement in sensorineural hearing loss after six months is rare, therefore final evaluation should be made one year after head injury.

GriffithsError: Reference source not found study showed a residual hearing loss in 14% of patients even after six months in cases of head injury with concussion without fracture.

VartianennError: Reference source not found reported that 1/3rd children recovered normal hearing with in a period of six months. Similar results were reported by ZimmermanError: Reference source not found.

DormanError: Reference source not found stated that in minor head injury, after excluding other causes of deafness, there is no sensorineural hearing loss of permanent nature. The hearing loss was noted to be due to cochlear dysfunction and temporary in duration.

According to Vernon and Press[54] only 8% of the patients who sustained head injury complained of tinnitus.

Dizziness is a frequent complication of head injury. Numerous studies have attempted to quantify the incidence of neuro-otological abnormalities in patients with post traumatic dizziness. Toglia [55]et al found out that 61% of patients had vestibular dysfunction. Ganon[56] et al reported 32% and Wilson[57] et al reported 57% neuro-otological test abnormalities in patients who sustained minor or moderate head injury. In case of recurring case of dizziness an organic etiology[58] must be suspected.

Hearing loss in the higher frequencies is sometimes seen as early as by age 20[59] [60]. It increases systematically to age 60 (and beyond) and is largest at 4 kHz and 6 kHz and is much larger in males than in females.

A small Sensorineural hearing loss of 25dB at the age of 25 has little medical or social relevance, however by the age of 70 a hearing loss as a result of aging is added to the pre-existing hearing loss. This results in a moderate to severe sensorineural hearing loss. In other words a seemingly minor hearing loss at a very young age may become severe when combined with other factors which affect hearing.

Figure-1, CROSSECTION OF THE EAR

Anatomy of the Ear and Temporal bone [61](Fig-1)

From the point of view of injury to the ear the anatomy can be divided into preauricular pinna, post auricular region, tympanic membrane, external auditory canal, middle ear and inner ear.

Preauricular region is the region of the ear in front of the auricle. Post auricular region is the region behind the auricle.

The temporal bone is a composite structure consists of tympanic bone, mastoid process, squamous and petrous parts. The tympanic bone forms the anterior, inferior and parts of the posterior wall of the external auditory canal. Laterally the tympanic bone borders the cartilaginous external auditory canal.

The sqamous portion of the temporal bone serves as the lateral wall of middle cranial fossa and interfaces with the parietal bone superiorly and with the zygomatic process and the sphenoid anteriorly.

The mastoid portion of temporal bone is the inferiorly extending projection seen on the lateral surface of the temporal bone. It is composed of a squamous portion laterally and petrous portion medially separated by korner's petro squamous septum. The petrous portion (Greek for 'rock like') guards the sensory organs of the inner ear.

For the purpose of description the ear is divided into four separate portions they are the auricle/ pinna, the external auditory canal, the middle ear and its derivative, and finally the inner ear.

The auricle (pinna) projects at an angle from side of head and has a role in collecting sound. The external auditory canal extends from the concha of the auricle to the tympanic membrane. It is partly bony and partly cartilaginous. The lateral cartilaginous portion is 8 mm long it is continuous with the auricular cartilage. The medial border of the meatal cartilage is attached to the rim of the bony canal by fibrous bands. The bony canal wall is 16mm long.

Tympanic bone constitutes the greater part of the canal; the squamous bone forms the roof. The tympanic cavity is an irregular, air -filled space within the temporal bone and contains the auditory ossicles and their attached muscles. The lateral wall of the tympanic cavity is part bony and part membranous. The tympanic membrane forms the central portion of the lateral wall, while above and below there is bone, forming the outer lateral walls of the epitympanum and hypotympanum respectively.

The tegmen tympani is the bony roof of the tympanic cavity, and separates it from the dura of middle cranial fossa. It is formed in part by petrous and part by the squamous bone, and the petrosquamous suture line, unossified in the young does not close until adult life.

The floor consists of a thin plate of bone which separates the tympanic cavity from the dome of the jugular bulb, sometimes it is deficient. The anterior wall of tympanic cavity is narrow as the medial and lateral walls converge. The lower portion of the anterior wall is larger than the upper and consists of a thin plate of bone covering the carotid artery.

The medial wall separates the tympanic cavity from the inner ear. Its surface possesses several prominent features and two openings. The promontory is the rounded elevation and occupying much of the central portion of the medial wall. It covers part of the basal coil of the cochlea and in front merges with the anterior wall of the tympanic cavity. Behind and above the promontory is the fenestra vestibuli (oval window) a nearly kidney - shaped opening which is closed by foot plate of stapes and its surrounding annular ligament. The fenestra cochleae (round window) lies below and a little behind the fenestra vestibuli. The membrane of fenestra cochleae does not lie at the end of the scala tympani but forms part of its floor. The scala tympani terminates posterior and medial to the membrane.

The inner ear or labyrinth lies in the temporal bone and for descriptive purpose is divided into a bony and a membranous portion. The bony labyrinth is derived from the inner periosteal layer of the otic capsule and consists of a thin, but dense, bony shell surrounding the vestibule, the semicircular canals and the cochlea.

The vestibule is the central portion of the bony labyrinth and is a small flattened ovoid chamber lying between the middle ear and fundus of internal auditory meatus. On its lateral wall is the opening of fenestra vestibuli which is closed by foot plate of stapes and its annular ligament. On the medial wall anteriorly is the spherical recess which contains the macula of the saccule.

Behind the spherical recess is the vestibular crest. Above and behind the crest is an elliptical recess which contains the macula of the utricle.The opening of the vestibular aqueduct lies below the elliptical recess, and the aqueduct itself passes through the temporal bone to open in the posterior cranial fossa but outside the dura.

The three semicircular canals, superior, posterior and lateral are situated above and behind the vestibule.

The bony cochlea lies in front of the vestibule and has an external appearance rather like the shell of a snail. The shell has approximately two and one half turns and its height is about 5mm while the greatest distance across the base is about 9 mm.

The coils of the cochlea turn about a central cone or modiolus which arises from the cochlear nerve portion of the fundus of the internal auditory meatus, and points laterally and forwards, tapering from a wide base to narrow apex.

The apex thus faces laterally and forwards towards the upper part of the medial wall of tympanic cavity, while the basal coil forms the bulge of the promontory below this. A thin shelf of bone arises from modiolus that spirals upwards within the lumen of the cochlea as the bony spiral lamina. A membrane - the membranous spiral lamina - extends from the edge of the bony spiral lamina to the outer wall of the cochlea, there by dividing each coil into the major portions, the scala vestibuli and scala tympani. Reissners membrane is a thin membrane stretching from the bony spiral laminae to upper part of the lateral wall of cochlear duct.

The endolymphatic surface consists of typical squamous epithelial cells with microvilli on their surface and joined together by tight junctions. Two types of epithelial cells have been recognized. The first type are flat and polygonal, Their surfaces being covered by short microvilli. The second type of epithelial cells are rounded in surfaces outline.

The first type are distributed throughout the Reissner's membrane and form a zone 2-3 cells wide at the limbic margin. The second type are arranged in distinct patterns along Reissner's membrane: bands, strands, whorls or clusters.

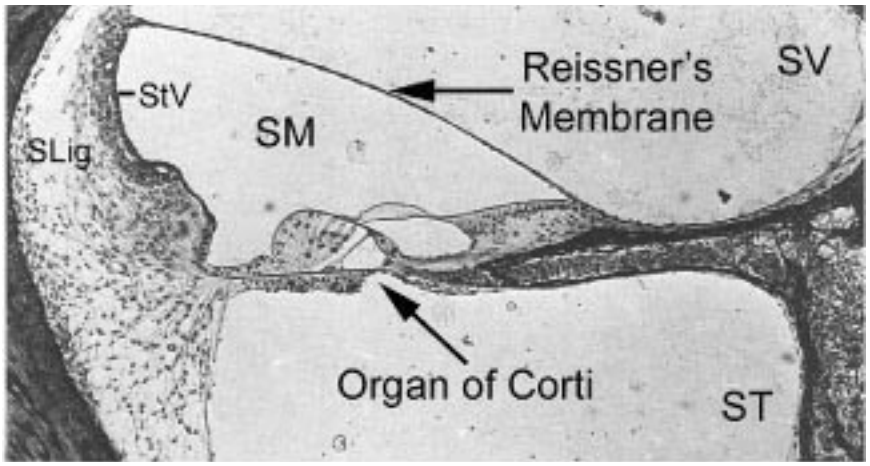
All the cells lining the scala media are joined by tight junctions which separate endolymph from the outside and maintain the ionic content. The tectorial membrane arises from the spiral limbus and extends over the organ of corti to attach close to the Hensen cell region.

The membrane is an acellular gel-like matrix containing fibrillar strands. The tips of the longest steriocilia of the outer hair cells are attached to or embedded in the undersurface of tectorial membrane and leave an impression on this surface.

The fluid space of the cochlea is divided length wise into three compartments called scalae, by the basilar membrane and the Reissner's membrane. The fluid space between the Reissner's membrane and the upper bony wall of the cochlea is the scala vestibuli. The fluid space between the basilar membrane and the lower bony wall is the scala tympani.

The space between the two membranes is called the scala media or the cochlear duct. Because it is bound on two sides by tissue membranes, the scala media is elastic and it responds to pressure from either side by moving in the appropriate direction.

The scala vestibuli and the scala tympani are filled with perilymph which is a typical extracellular fluid rich in sodium and low in potassium.The scala media contains endolymph which is a unique extracellular fluid low in sodium and high in potassium.



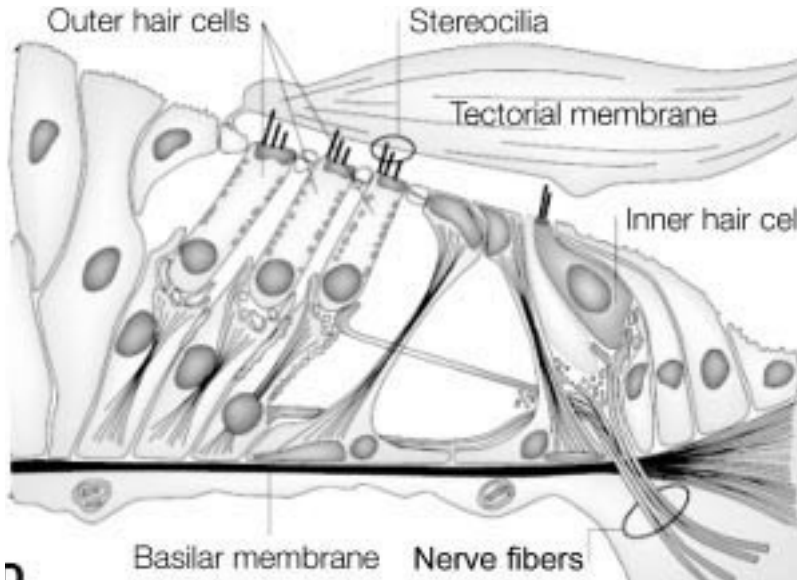


Fig-2, CROSS SECTION OF COCHLEA

THE COCHLEA (Fig-2**)**

The basilar membrane which separates the scala media from the scala tympani consists of connective tissue fibers embedded in an acellular matrix. The organ of corti, tectorial membrane along with the basilar membrane makes up the cochlear partition.

The organ of corti is a ridge like structure containing the auditory sensory cells and a complex arrangement of supporting cells. The sensory cells are arranged in two distinct groups as inner and outer hair cells. There is a single row of inner hair cells, although occasionally extra hair cells may be apparent, and also three, four or five irregular rows of outer hair cells, with frequent gaps where individual hair cells are absent. Each hair cell consists of a body, which lies with in the organ of corti, and a thickened upper surface called the cuticular plate, from which projects a cluster of steriocilia or hairs.

The steriocilia contains a core of actin molecules packed in a para crystalline array and covered with a cell membrane. The steriocilia are connected to each other along the sides by fine filaments called the side links. The tip of each stereocilium is connected to the sides of the next tallest steriocilium by a longer filament known as a tip link.

The body of the inner hair cells is flask shaped, with a small apex and large cell body. The long axis of cell is inclined towards the tunnel of corti, and nerve fibers and nerve endings are located around the lower half of the body. The steriocilia projecting from the thickened cuticular plate are arranged in two or three rows parallel to the axis of the cochlear duct.

The body of the outer hair cell is cylindrical with the nucleus lying close to the lower pole, where afferent and efferent nerve endings are attached. There are several rows of steriocilia but the configuration varies from a W shape at the base, through a V shape in the middle coil, to almost a linear array at the apex. The number of steriocilia also decreases in the passage from base to apex, where as the length increases, although not in a linear fashion.

The hair cells are supported with in the organ of corti by several types of specialized, highly differentiated cells. These are the pillar cells, Dieters cells and Hensen's cells.

In the fetus and the newborn there are about 3500 inner hair cells and 13000 outer hair cells.

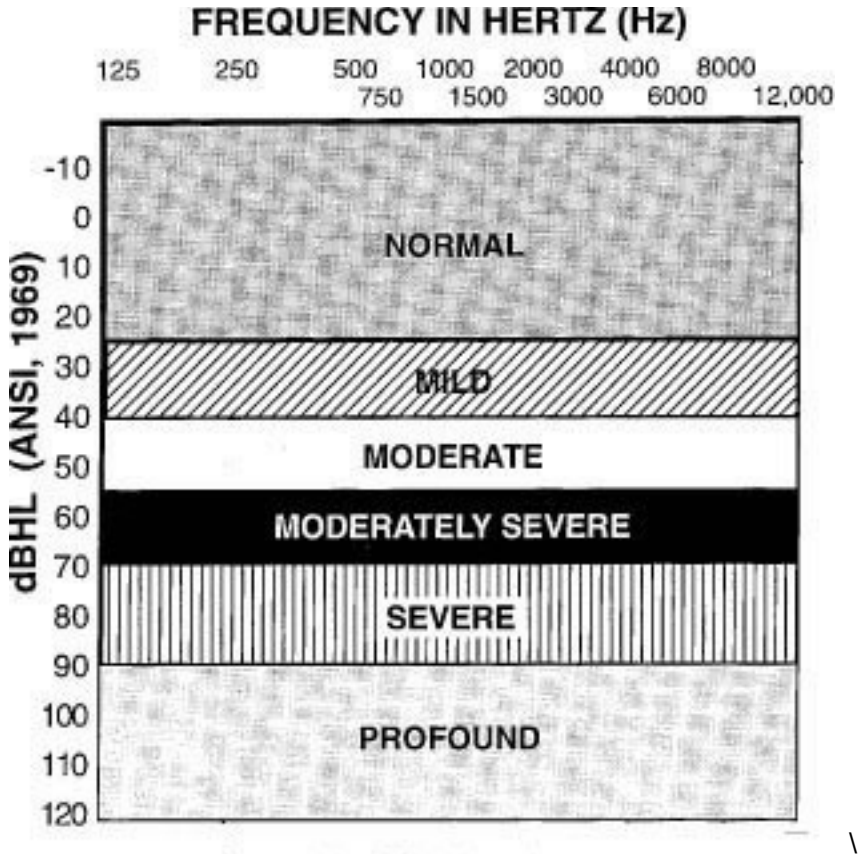


Fig-3, CLASSIFICATION OF HEARING LOSS

AUDIOMETRIC TESTS

Pure Tone Audiometry:

It is the most commonly used method of measuring hearing acuity. It is a subjective test. The frequencies usually tested are at octave steps i.e, 125, 250, 500, 1000, 2000, 4000, 8000 Hz.(Fig-3) A pure tone audiometer is an electronic instrument capable of producing pure tone sound of different frequencies at variable intensities. It helps in qualitative and quantitative diagnosis of hearing loss.

TYMPANOMETRY is the measurement of acoustic emissions in the external auditory meatus as a function of air pressure within the external auditory meatus. It provides a rapid atraumatic and objective technique for evaluating the integrity of

1. Middle ear transmission system

2. Estimating middle ear pressure

3. Estimating volume of ear canal or middle ear

4. Evaluating Eustachian tube function

Metz was the first to apply the concept of acoustic impedance to clinical audiology.

Acoustic impedance is a measure of apposition to flow of acoustic energy into the middle ear transmission system. The first tympanograms were reported by Terkildsen and Thomsen in 1959.

One of the more commonly used procedures is classification of tympanograms according to shapes. It was first described by Liden and modified by Jerger[62] and others.

Type A Tympanogram indicate normal middle ear pressure as indicated by tympanogram peak at 0 daPa. Normally middle ear pressure typically falls between +50 and –100 daPa.

The volume of the ear canal can also be estimated. In general, ear canal volumes normally range from 0.5 to 1.0 ml in children and 0.6 to 2.0 ml in adults.

Volume measurements more than 2 ml in children and 2.5ml in adults are usually indicative of tympanic membrane perforation or patent pressure equalization tube.

ACOUSTIC REFLEX MEASURES IN AUDIOLOGIC EVALUATION

The first observation of the acoustically evoked contraction of the middle ear muscles were made by Hensen who observed the responses of the tensor tympani and stapedius muscles to sound stimulation in dogs.

Luscher first reported the acoustic reflex in humans by observing the stapedius tendon through a perforated tympanic membrane.

The first electro acoustic device used for clinical acoustic reflex measurements was developed by Metz called the Metz bridge. The effects of cochlear hearing loss on Acoustic reflex measurements.

Mild cochlear hearing loss has little effect on acoustic reflex thresholds for tonal stimuli.

As the hearing loss increases above 40 dB, acoustic reflex thresholds increase approximately linearly and for patients with hearing losses that exceed 70 dB reflexes are typically absent.

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OTO ACOUSTIC EMISSIONS:

The origins of the understanding of how the cochlea processes sounds are in the mid 19th century. At that time the frontier of science included the theory of music and musical instruments. Mathematicians such as Fourier had succeeded in interpreting complex sounds as the summation of simple generic sounds or harmonics.

With the perfection of the compound microscope scientists such as Helmholtz[63] thought that frequency specific resonators in the ear were in the fibers of organ of corti and basilar membrane and the place theory of hearing was born.

In 1930s Wever and Bray[64] identified the cochlear microphonic signal, which supported the telephone theory of hearing.

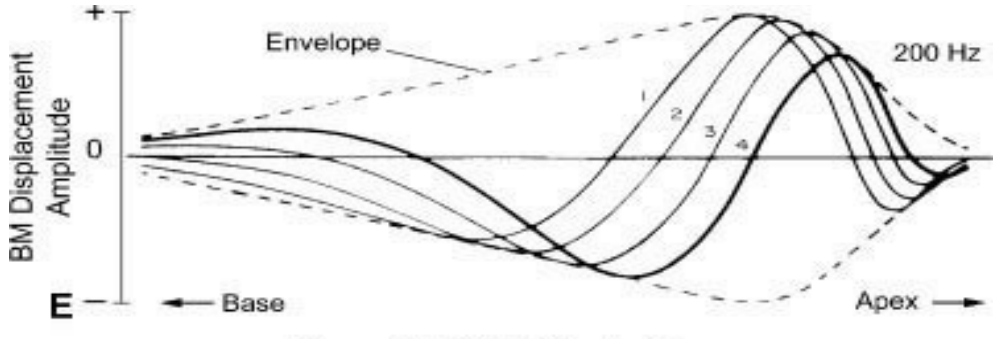


Fig-4, BASILAR MEMBRANE DISPLACEMENTS PRODUCED IN CADAVERIC HUMAN COCHLEA IN RESPONSE TO 200Hz AT 4 SEPARATE POINTS OF TIME.ENVELOPE OF TRAVELLING WAVE IS ALSO NOTED

Von-bekesy[65] (Fig-4) discovered that a relatively slow travelling wave was created that neatly delivered incoming sound energy of different frequencies to different places along organ of corti.

As early as 1948, GOLD[66] discovered that the outer hair cells of the cochlea could produce energy by an active mechanical process. However it was not until 1978 that Kemp[67] by a series of basic and clinical experiments demonstrated that the cochlea was capable of producing low intensity recordable sounds called oto-acoustic emissions.

Oto-acoustic emissions (OAEs) can be defined as the audio frequency energy which originates in and is released from the cochlea, transmitted through the ossicular chain and tympanic membrane and measured in the external auditory meatus. They can occur either spontaneous or in response to acoustic stimulation.

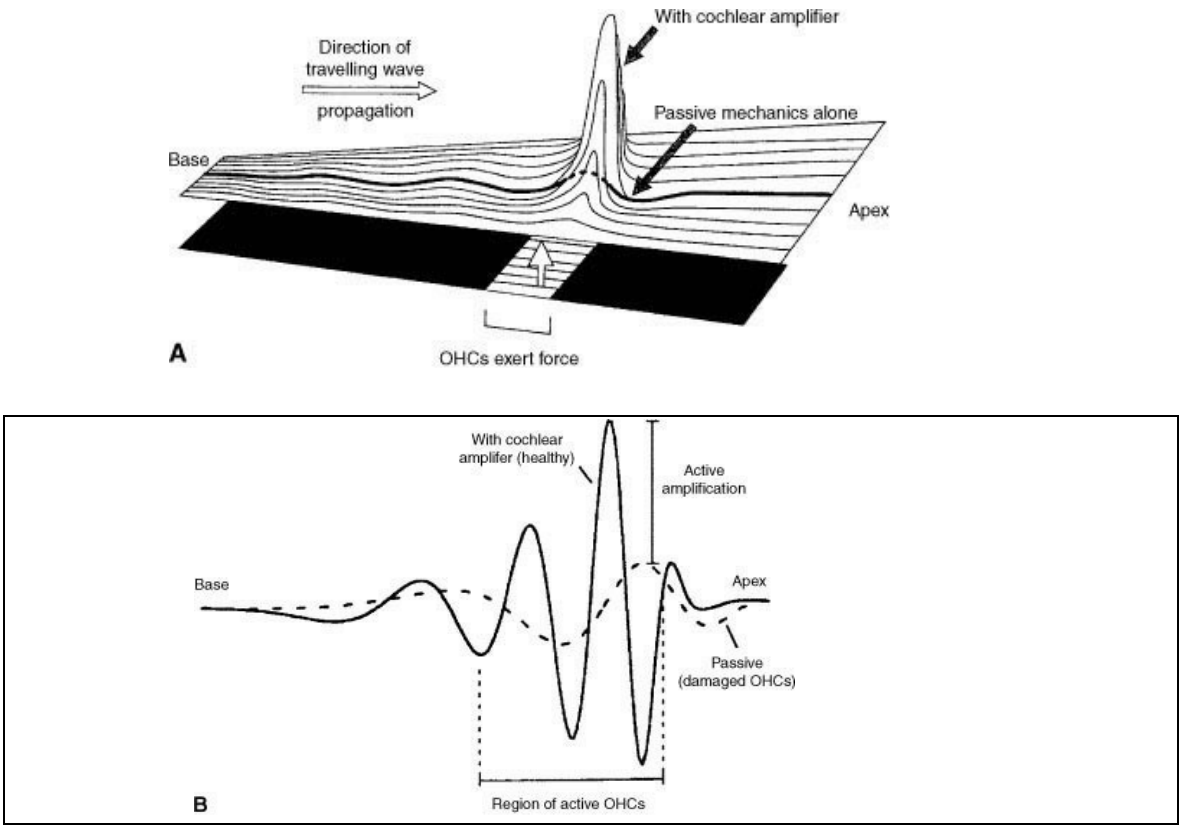
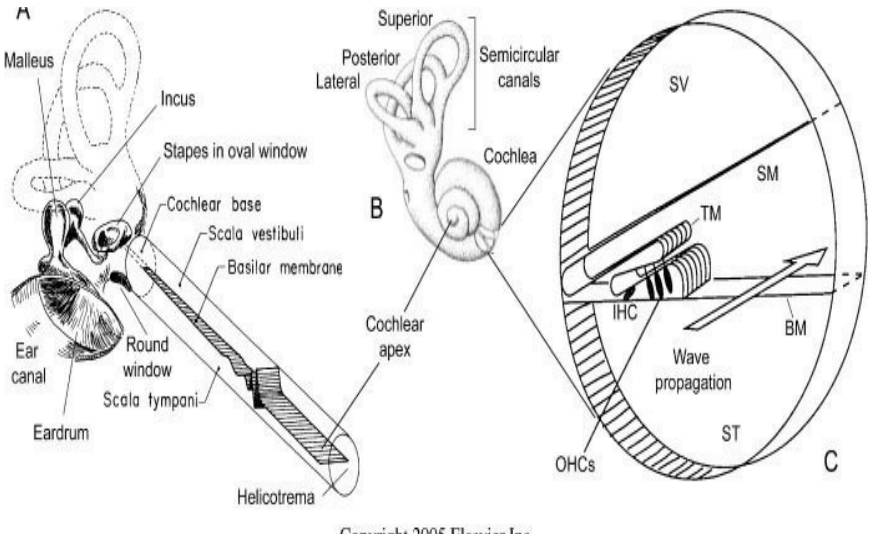


Fig – 5, SCHEMATIC REPRESENTATION OF TRAVELLING WAVE ALONG BASEMENT MEMBRANE

OAEs are believed to reflect the active biomechanical movement of the basilar membrane of the cochlea (.Fig-5). This retrograde travelling wave is thought to be responsible for the sensitivity, frequency selectivity and wide dynamic range of the normal auditory system.

Oto-acoustic emissions (OAEs) are believed to be the by product of pre-neural mechanisms of the cochlear amplifier and in particular, to be linked to the normal functioning of the outer hair cells. Oto-acoustic emissions are vulnerable to a variety of agents such as acoustic trauma[68], hypoxia[69], and oto-toxic[70] medications that cause hearing loss by damaging outer hair cells.

Taking into account estimates of amplification provided by outer hair cells, complete destruction of OHC'S alone could result theoretically, in a hearing loss of 60 dB. Early investigations in to OAE'S proved that they are not present when the sensorineural hearing loss exceeds 40-50dB [71,72].



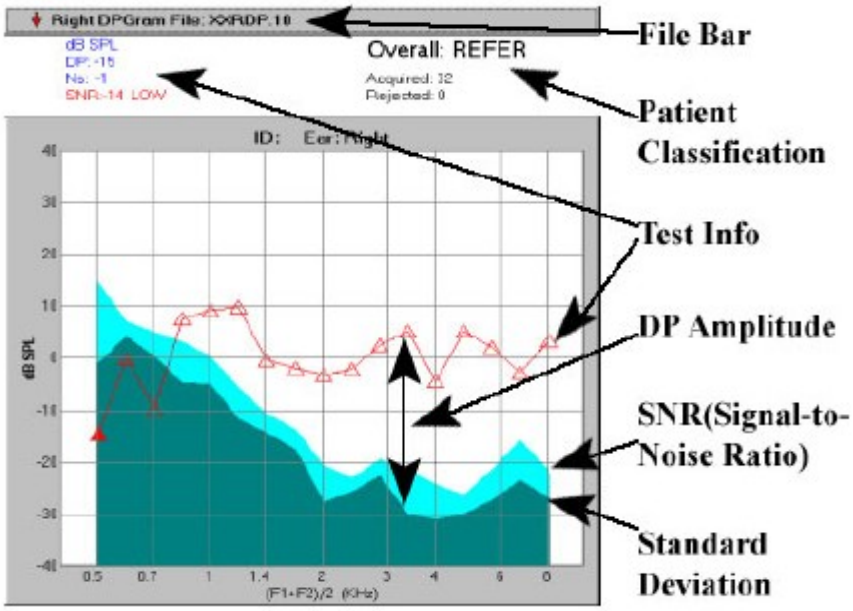


Fig-6, SCHEMATIC REPRESENTATION OF THE TRAVELLING WAVE IN COCHLEA WITH DPOAE GRAM

CLASSIFICATION

OAEs(Fig-6) - are classified as either spontaneous or evoked. Spontaneous occur in the absence of any exogenous stimulation, and evoked oto-acoustic emissions occur in response to a presentation of acoustic stimulus. Distortion product oto-acoustic emissions are evoked OAEs which are generated by two continuous pure tones separated by a specific frequency difference. Evoked OAEs can be detected in essentially all ears with normal hearing and are reduced or absent in ears affected by cochlear disorders.

DPOAEs measures have shown excellent intra-subject test reliability which allows monitoring of dynamic changes of cochlear function[73].

DPOAEs are measured in response to two tones presented to the ear. The interaction of the two tones presented gives rise to an audible signal at a specific additional frequency. Most but not all distortion product energy is generated in and emitted directly from the f2 emission site of the basement membrane. The primary tones can therefore be selected to test a specific frequency region. By convention ,the lower frequency pure tone is referred to as f_1 primary, and its level as L_1 .The higher frequency pure tone is referred to as f_2 primary and its level as L_2 .DPOAEs are widely believed to be a rapid objective, reliable and repeatable measure of the physiological integrity of outer hair cells of cochlea.

The amplitude of DPOAE is also systematically dependent on the relative levels of the stimulus tones and can be increased slightly for suprathreshold stimuli by decreasing L_2 below L_1[74,75].

DPOAE level is dependent on the frequency separation of the stimulus tones, being largest Error: Reference source not foundwhen f_2/f_1 is around 1.21 to 1.22. It has been established that DPOAEs are reduced or eliminated by compromise of middle ear conduction pathway (Fig 7). Normal middle ear functioning is pre-requisite for measuring DPOAE and it is therefore important to include immitance measurements while recording DPOAEs. This means is also used to confirm the presence of any middle ear pathology.

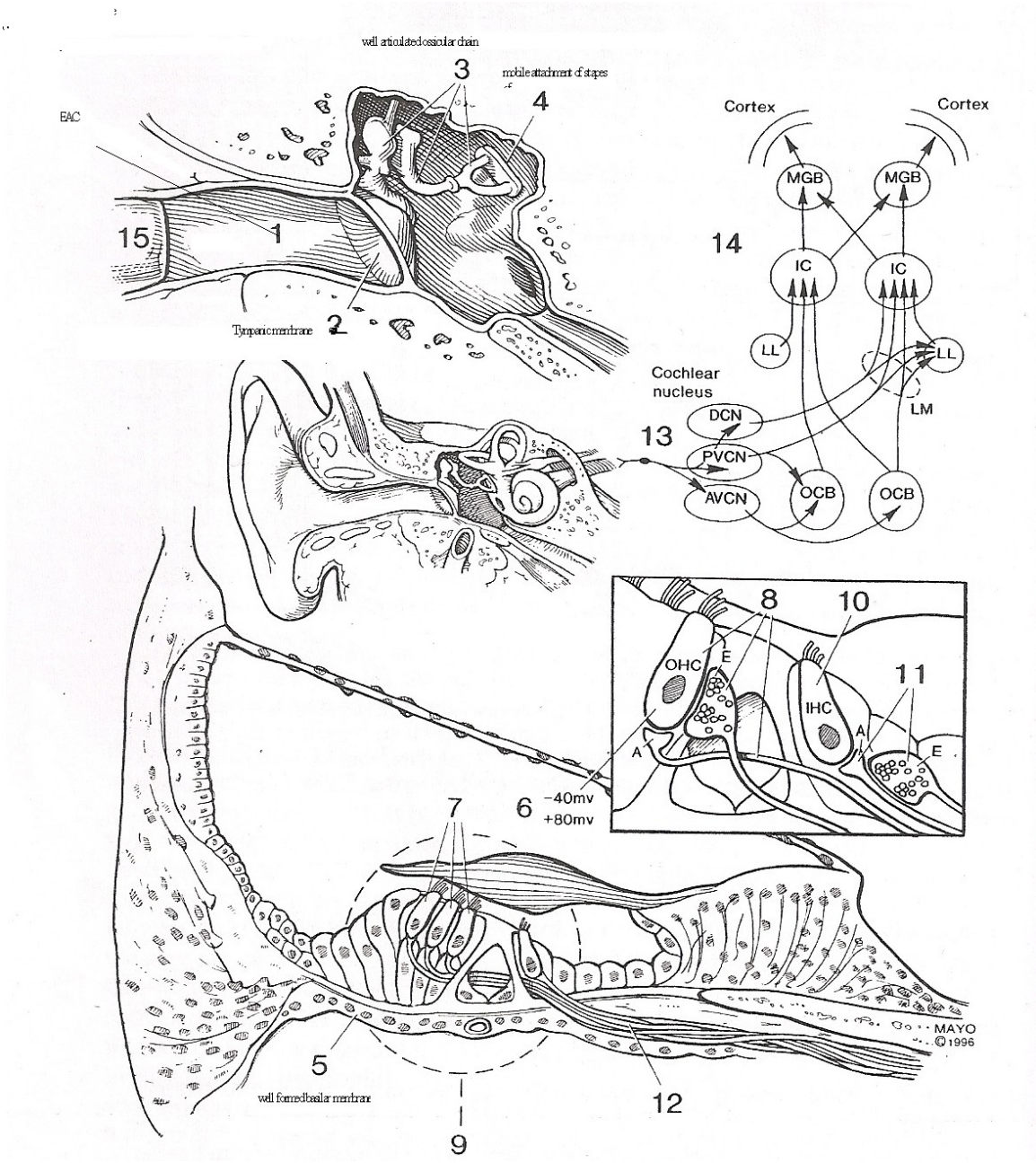


Fig-7 SCHEMATIC DIAGRAM LISTING FACTORS ASSISTING AUDITORY THRESHOLD

PATIENTS AND METHODS

This prospective study was done in the ENT department of Christian Medical College Vellore. A pilot study was done initially for 3 months to evaluate hearing loss in patients presenting to the Accident and Emergency department with history suggestive of mild head injury. Based on the pilot study the sample size was determined for the main study.

60 patients with history suggestive of mild head injury were recruited in to the study which was conducted over a period of six months and they were followed up for six months. Each patient was examined in emergency department by casualty medical officer. A thorough neurological examination was done and the patient was assigned a score according to the Glasgow coma scaleError: Reference source not found. A thorough neuro-otological examination was done by an ENT surgeon in the emergency department.

Once the inclusion criteria were fulfilled the patient was included in the study and was followed up. An informed consent was taken and a proforma was filled.

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INCLUSION CRITERIA:

Head Injury.

GCS 13 – 15 and improving.

Age between 6 – 60 years.

Patients discharged from casualty after observation period of 24 hours.

History of loss of consciousness of less than 20 minutes.

EXCLUSION CRITERIA:

1. Patients with past history of ear disease, previous head injury or noise trauma.
2. Patients having family history suggestive of autoimmune disease and hearing loss.

METHODOLOGY

A pilot study was conducted for a period of three months from September2006 to December 2006, to determine the procedures of data collection. Twenty patients were recruited into the pilot study. Of these 16 patients were found to have hearing loss in the immediate post trauma period. The sample size for this prospective study was calculated based on the pilot study.

Patients in the age group of 6 to 60 years were evaluated. 60 patients with history suggestive of mild head injury were included in the study. A detailed evaluation of the severity of injury using Glasgow coma scale scoring was done. Radiological investigations like X-Ray skull (antero-posterior and lateral which are routinely done) and CT scan were used to detect skull fractures.

Blood investigations – Hemoglobin, serum electrolytes, random blood sugar, and creatinine were done and vital signs were monitored through out the observation period. Patients with deterioration of GCS score were evaluated with CT scan. A detailed neuro-otological examination was done. Patients were specifically asked for symptoms of hearing loss, tinnitus and vertigo.

The external auditory canal and tympanic membrane were assessed to rule out any signs of temporal bone fracture like bleeding from external auditory canal, palpable step deformity, tympanic membrane perforation or haemotympanum. If the external auditory canal was filled with clotted blood patient was called for assessment after a period of one week. Eyes were checked for nystagmus and conjugate deviation. Facial nerve function tests were done and when the patient was cooperative facial nerve function was graded according to House Brackmann scale[76]. Tuning fork tests were done if the patient was cooperative.

Patient was called to audiology lab and a detailed hearing assessment was done.

PURE TONE AUDIOMETRY

Pure tone audiometry was done using GSI-61, MA-53 audiometers. They were calibrated daily with subjective calibrations with normals. Hearing thresholds of 15-25 dB across the frequencies were considered to be as normal.

A Siemens SD-30 impedance audiometer was used to obtain immitance measurements. The test was performed using a probe tone frequency of 226Hz

An ipsilateral stapedial reflex at 1000 Hz was elicited. The ipsilateral acoustic reflex threshold was seen as normal if the level at which it is elicited falls between 70db and 100db.

OTO-ACOUSTIC EMISSION TESTING

DPOAE testing was done with the Smart DPOAE package of INTELLIGENT HEARING SYSTEMS. DPOAEs were recorded using a test protocol where primaries were fixed at L_1=65dB SPL, L_2=55dB SPL with an f_2/f_1 ratio of 1.22. The f_2 frequencies were carefully selected to correspond closely to audiometric test frequencies of 1000 Hz, 2000 Hz, 4000 Hz

DPOAE testing was done at1000Hz, 2000Hz, 3000Hz, 4000Hz, and 6000Hz. A DPOAE response was considered to be present if the SNR >6.13dB.

The DPOAES were recorded with DPOAE amplitude in dB SPL as a function of stimulus frequency. This is commonly known as DPOAE gram. The f2 frequencies are presented on the horizontal axis and the amplitude of the DPOAE for the different frequencies are plotted on the vertical axis

Repeat evaluation was done after a period of three and six months. A detailed oto-neurological evaluation was done in all three visits and patients were specifically asked for symptoms of hearing loss tinnitus and vertigo.

Letter‘s were sent to patients who did not come for repeat auditory assessment.

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RESULTS AND ANALYSIS

Of the 60 patients, 7 patients were found to have normal audiograms and presence of DPOAEs. 18 patients came for regular follow up till six months, 16 patients came for follow up for a period of three months. Of these patients 5 came for follow up after six months. 19 patients did not come for follow up.

The following observations were made from the study. Statistical analysis was done using SPSS version 11.0. Variables were assessed using Wilcoxon Signed Ranks Test and McNemar Test

AGE AND SEX DISTRIBUTION: **TABLE-I**

AGE IN YEARS	MALE	FEMALE	TOTAL
0 – 10	0	2	2
11 – 20	7	2	9
21 – 30	15	0	15
31 – 40	14	3	17
41 – 50	11	1	12
51 – 60	3	2	5

TOTAL	50	10	60

83% were males. 66% were between ages of 20-50 years (Fig.8 & 9)

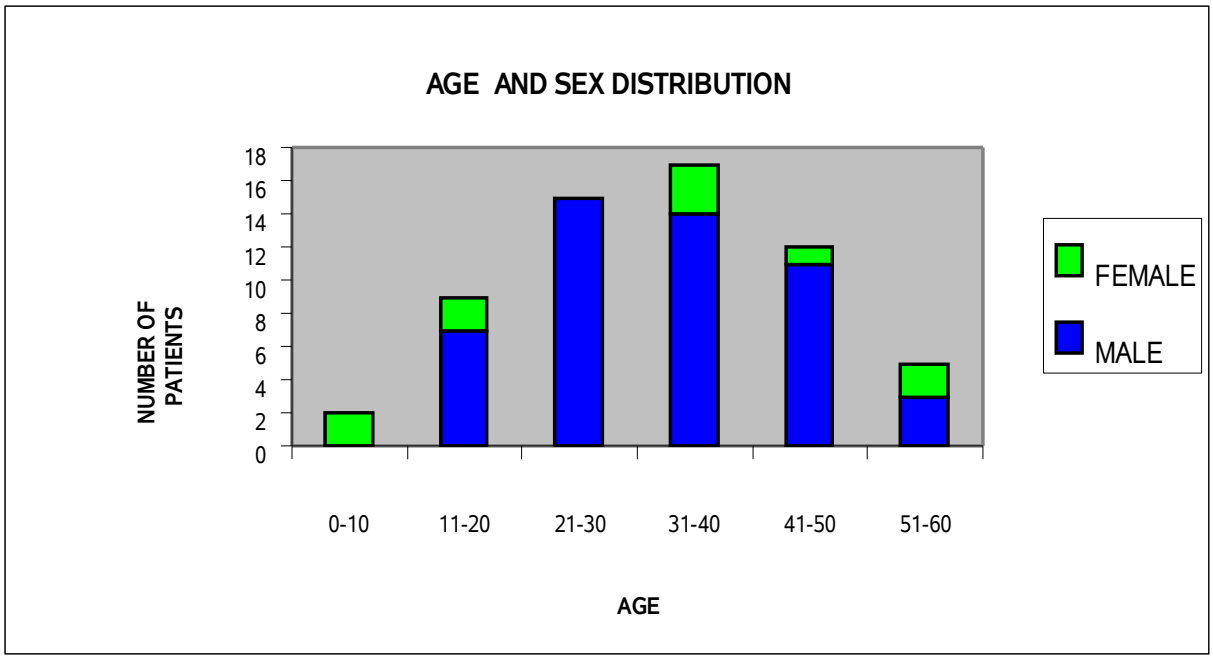


Figure-8

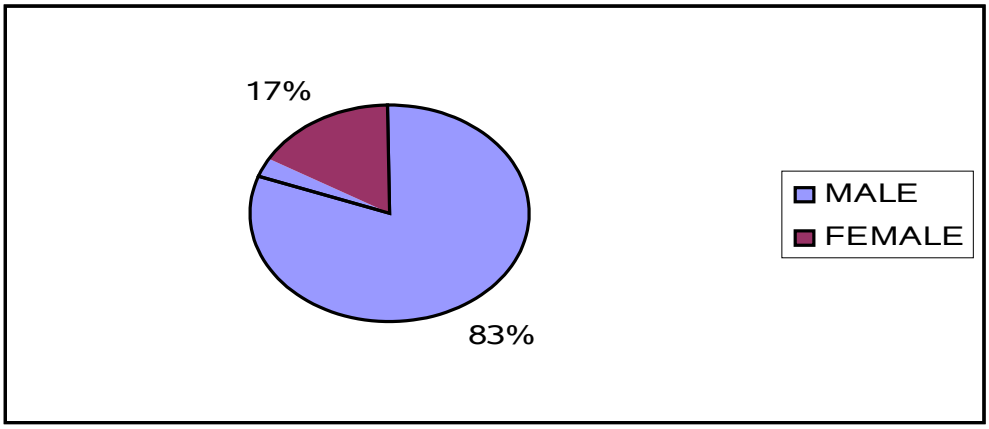


Figure-9

MODE OF INJURY

TABLE-II

ROAD TRAFFIC ACCIDENTS	FALL FROM HEIGHT	HIT BY A BRICK	BULL GORE INJURY

54	4	1	1

Road traffic accidents were the major cause of Head injury (Fig.10)

SYMPTOMS

TABLE-III

ASYMPTOMATIC	HEARING LOSS	VERTIGO	TINNITUS
44	6	9	1

Out of 60 patients 73% were asymptomatic, 15% complained of vertigo, 10% complained of hearing loss, and 2% complained of tinnitus. (Fig.11)

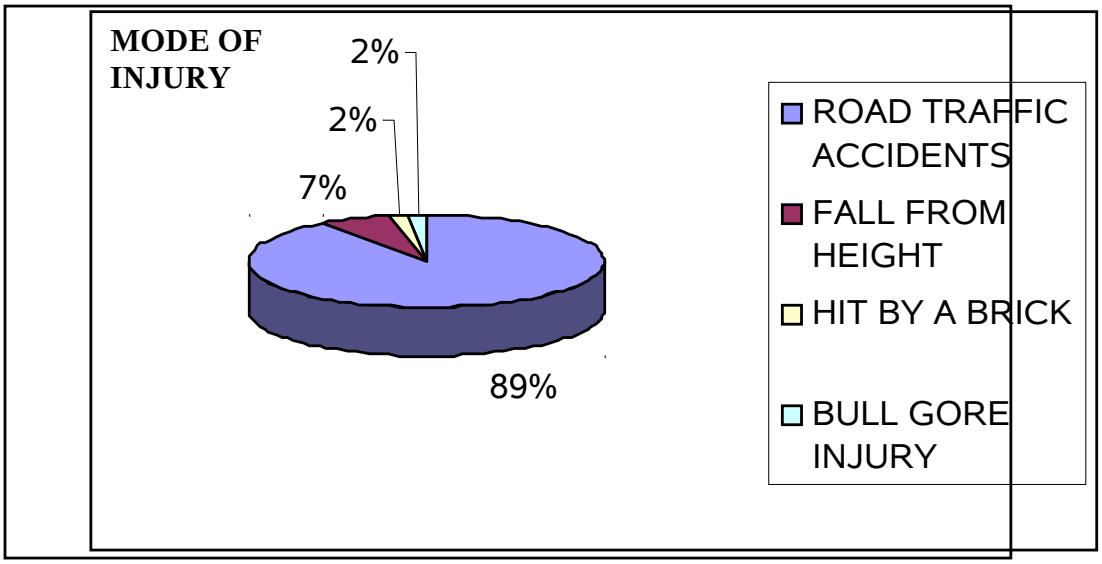


FIGURE-10

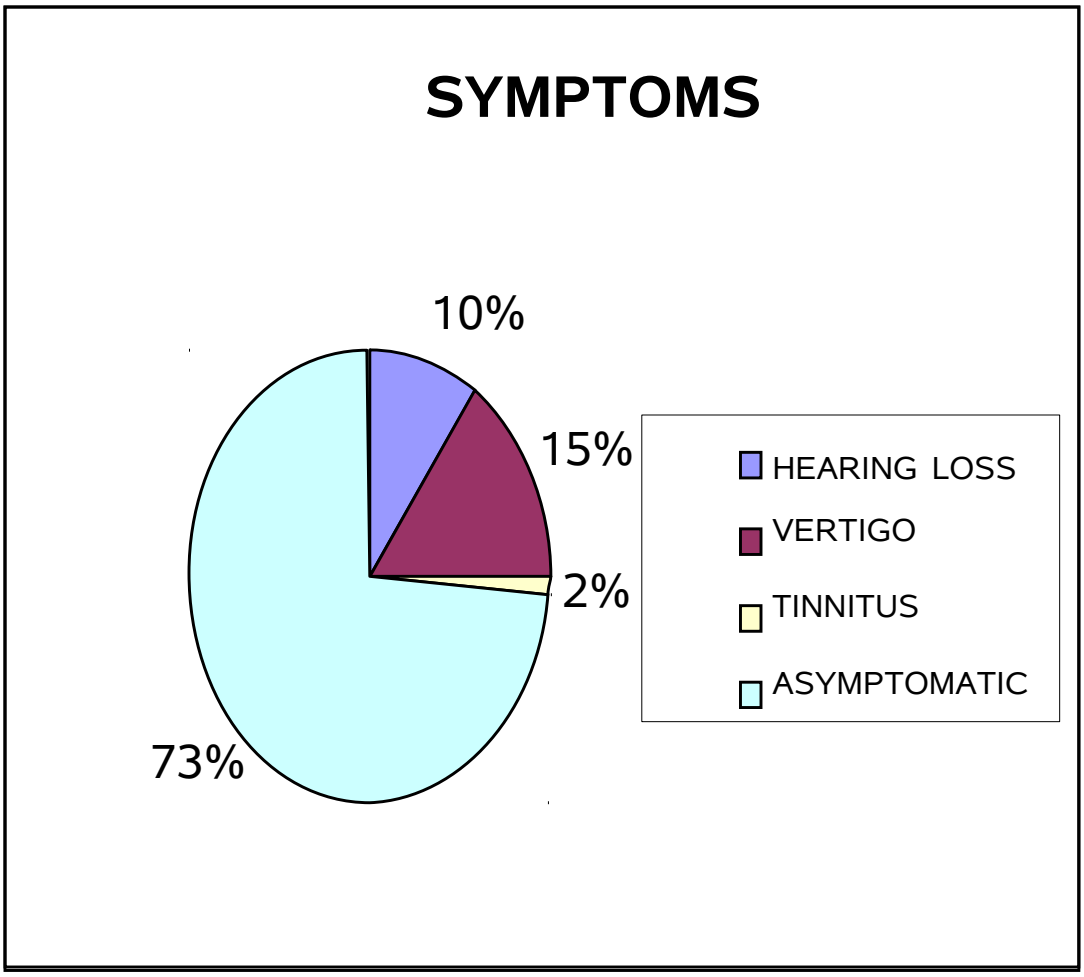


FIGURE-11

HEARING LOSS ON EVALUATION

TABLE-IV

NORMAL	SNHL	CHL	MIXED
46	49	8	17

Out of 60 patients (120 ears tested) 38 % had normal hearing, 40% had sensorineural hearing loss, 7% had conductive hearing loss, and 15 % had mixed hearing loss. (Fig.12)

Pure tone audiometry assessment of hearing immediately post trauma with respect to frequencies affected revealed that hearing loss was mainly in the high frequency region with greatest loss noticed at 4000 Hz and 8000 Hz. (Fig.13)

Significant hearing improvement was found at all the four frequencies with in three months 1000Hz (p-value 0.014) (Fig .14), 2000Hz (p-value 0.006)(Fig.15), 4000Hz (p-value<0.001)(Fig .16), 8000Hz (p-value 0.002)(Fig.17).

Significant improvement in DPOAEs was noted in all the four frequencies with in three months, 1000 Hz (p-value 0.012)(Fig .18) , 2000Hz(p-value 0.041)(Fig.19), 3000Hz (p- value 0.002)(Fig.20) , 4000Hz (p- value 0.001)(Fig .21) , 6000Hz(p- value0.013)(Fig.22).

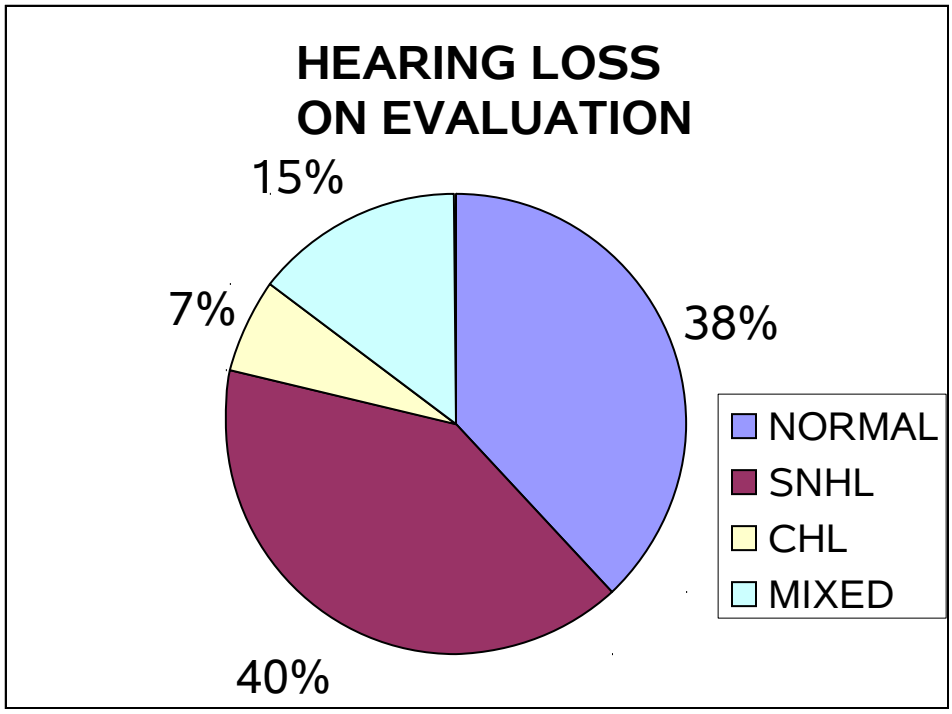


FIGURE-12

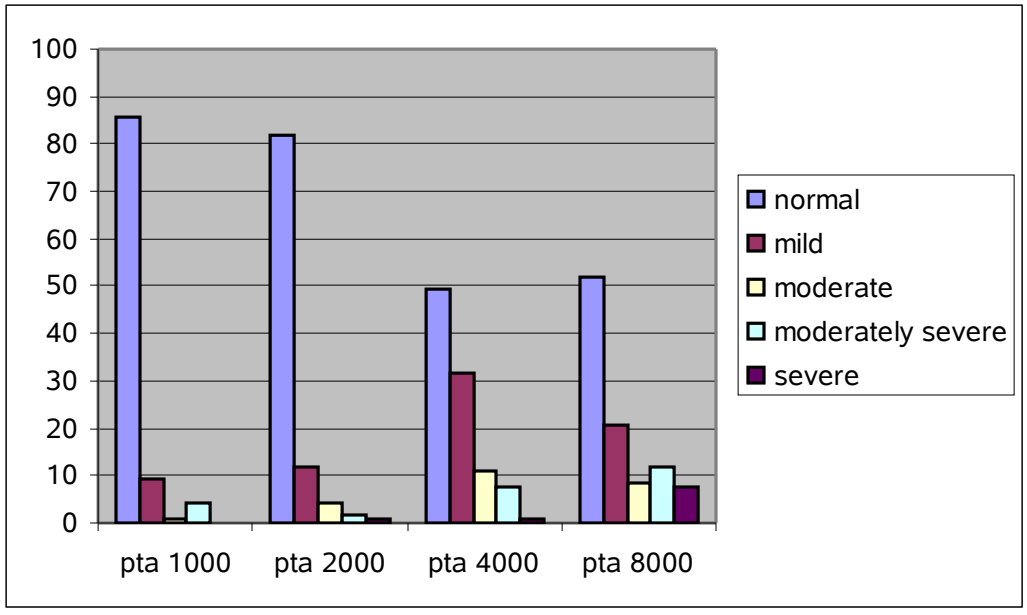


FIGURE-13

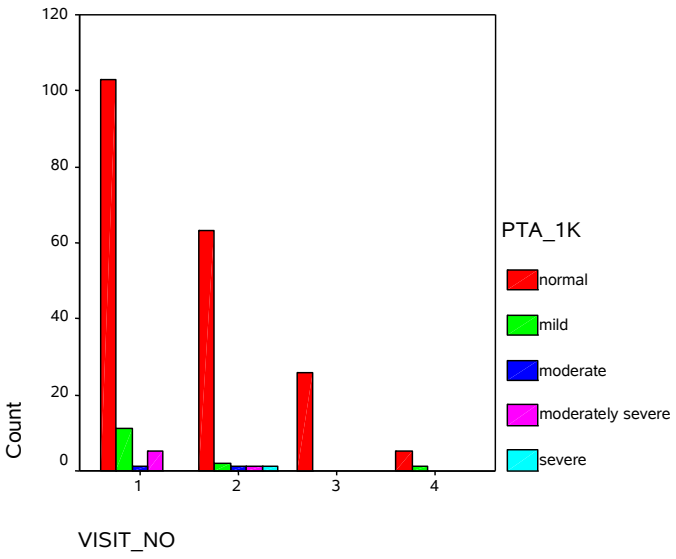
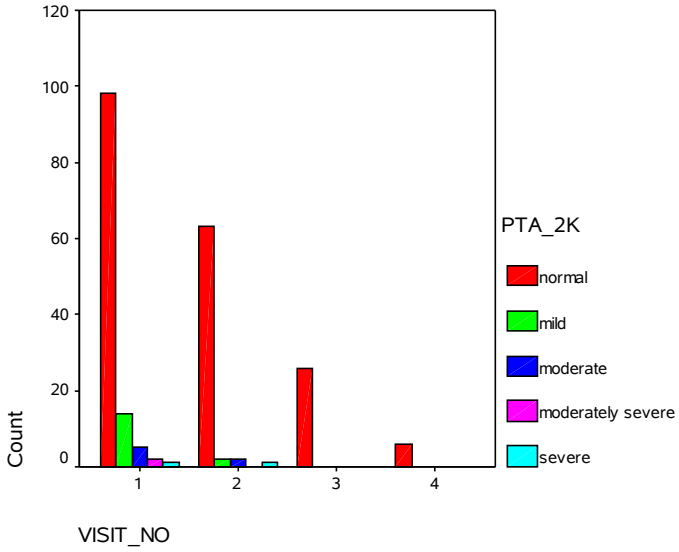


Figure-14



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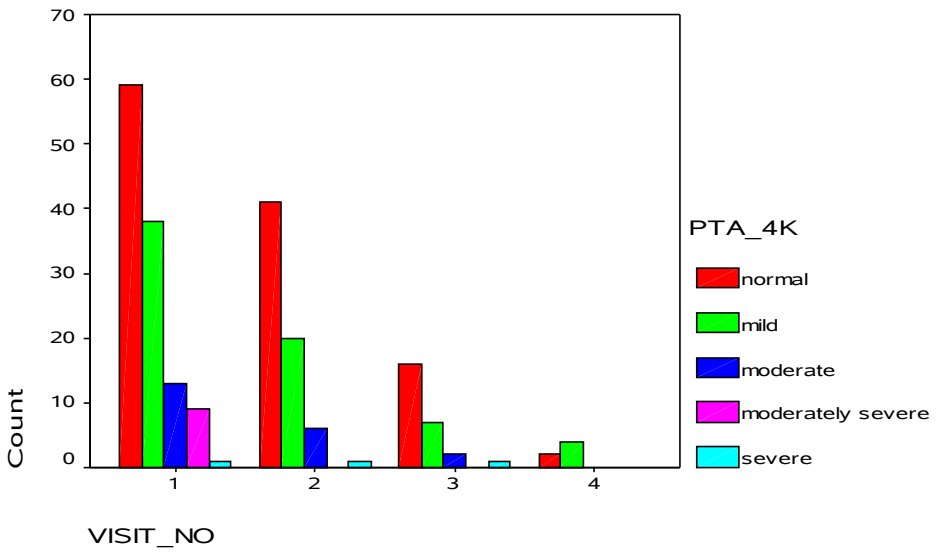

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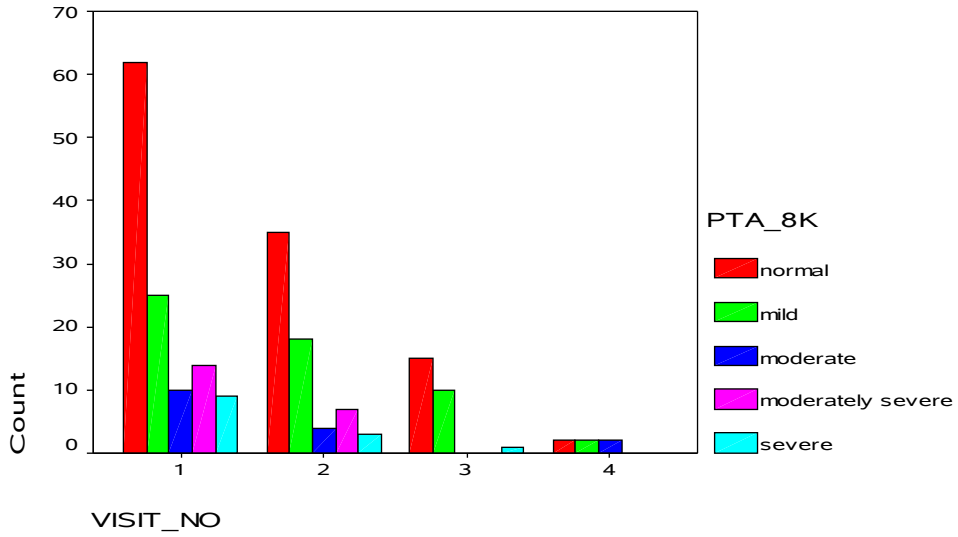

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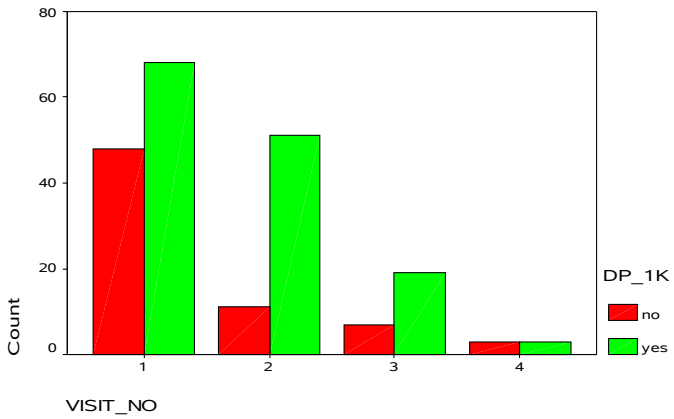

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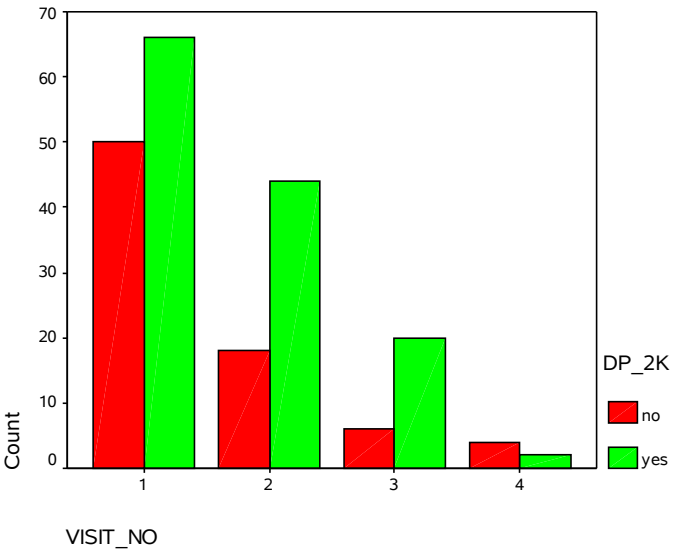
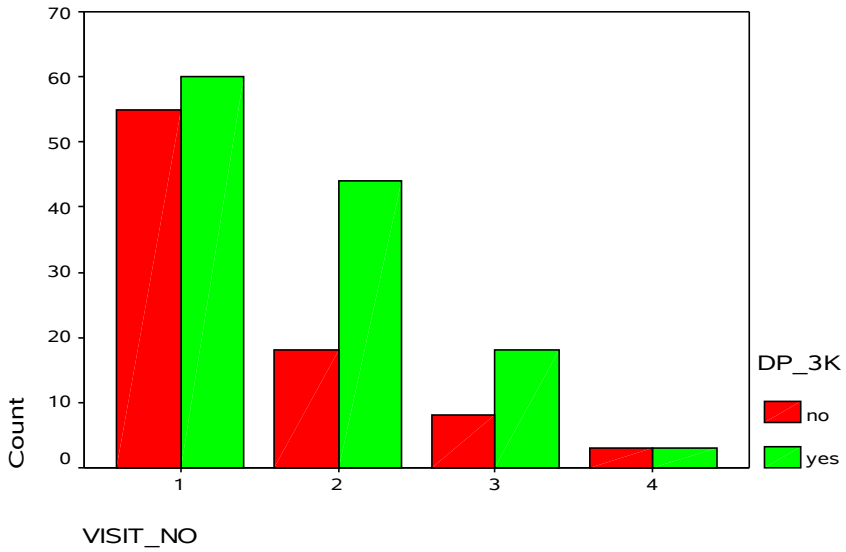


FIGURE-19



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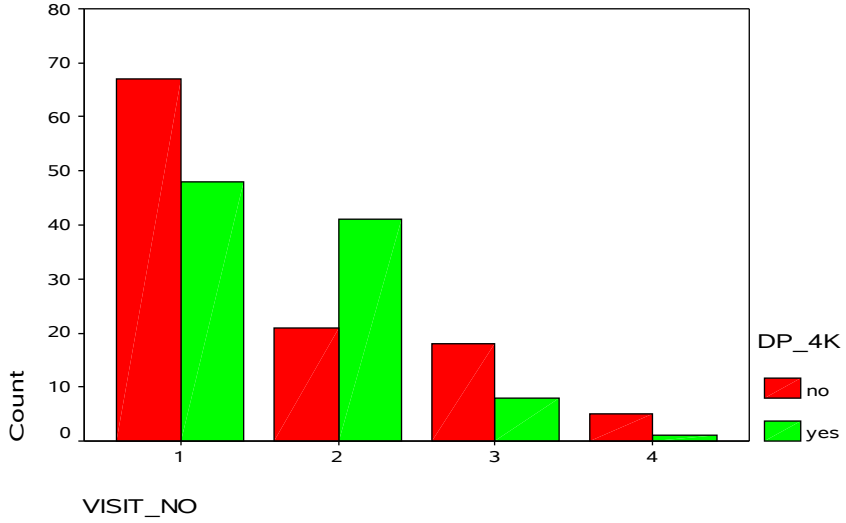

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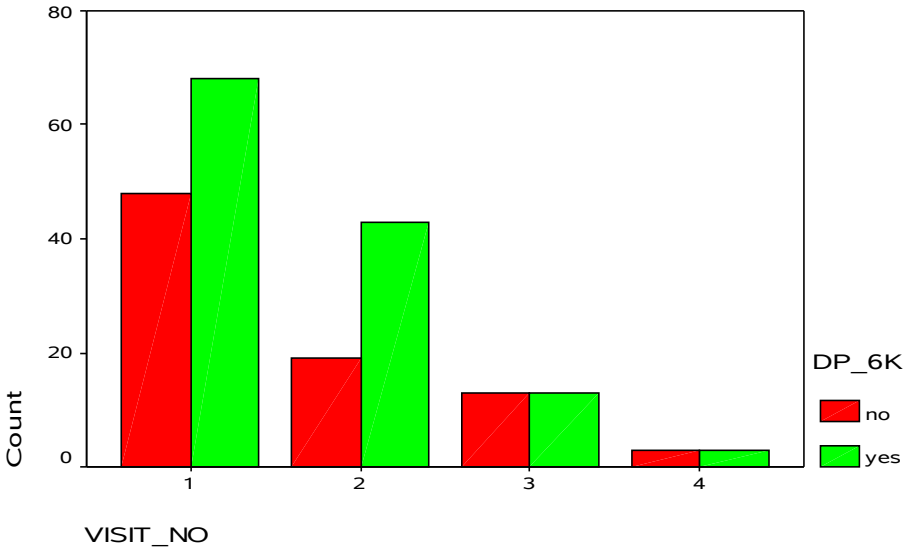

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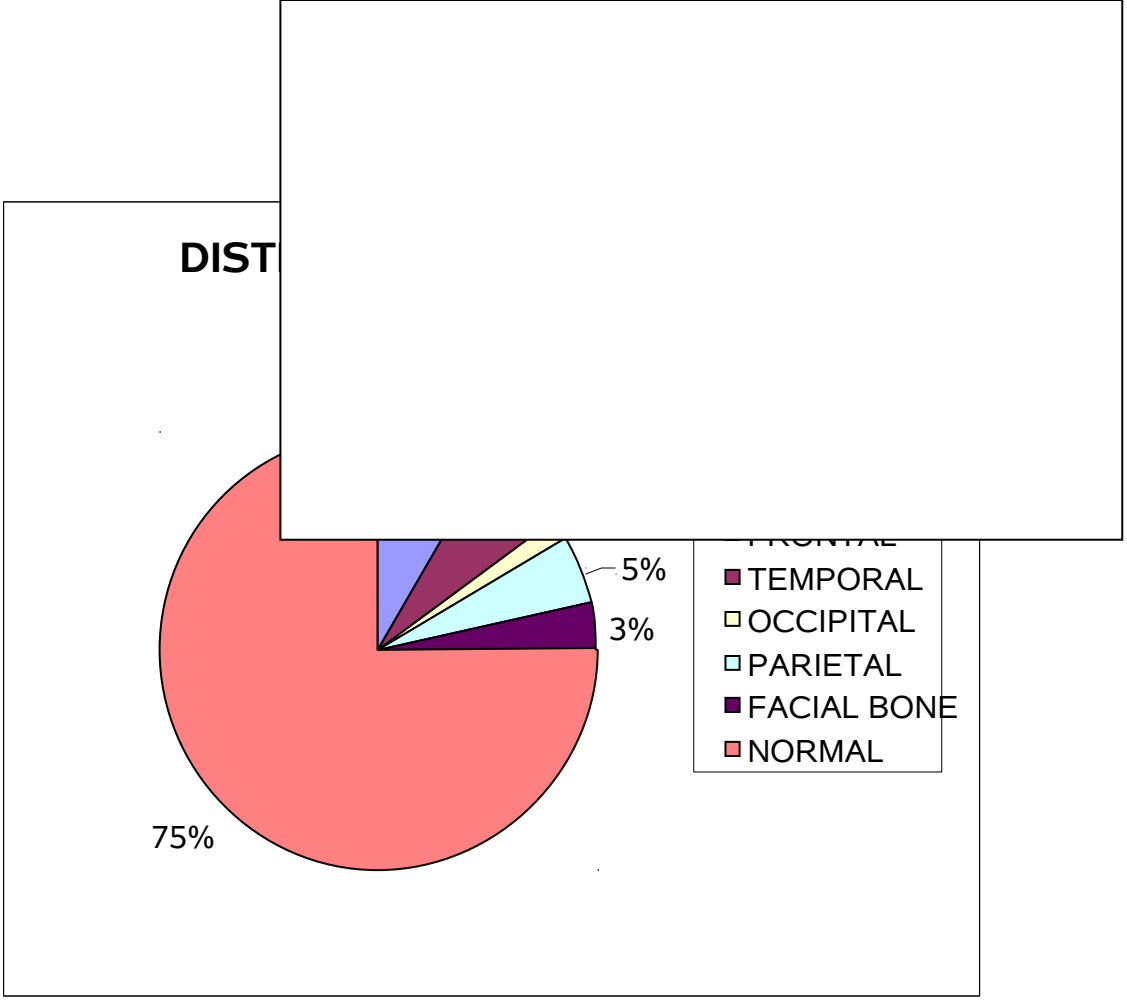

FIGURE-23

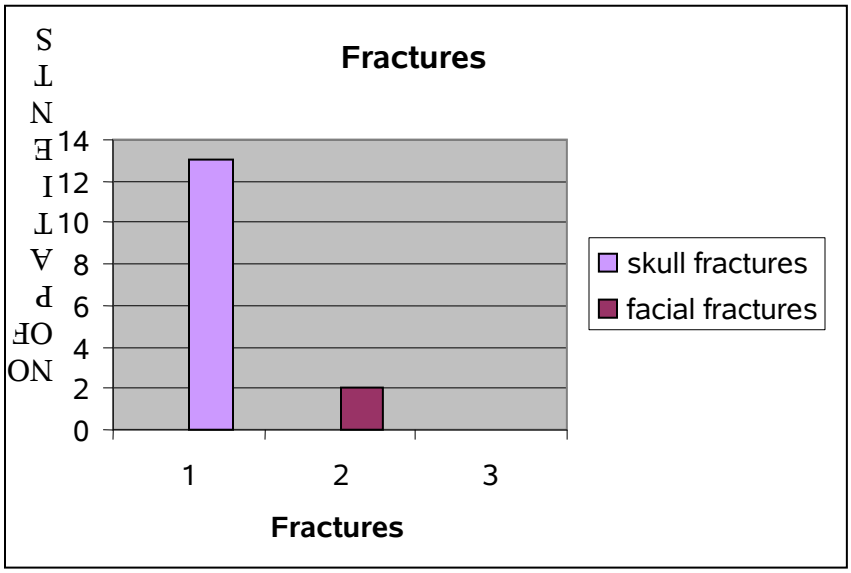

FIGURE-24

CLASSIFICATION OF FRACTURES

TABLE V (Fig.23)

NORMAL	FRONTAL	TEMPORAL	OCCIPITAL	PARIETAL	FACIAL BONE

45	5	4	1	3	2

Out of 60 patients a total number of 15 patients were found to have skull fractures. (Fig.24)

Of 5 patients with frontal bone fractures bilateral hearing loss was noticed in all the patients, of which mixed hearing loss improved but did not become normal.

In patients who sustained temporal bone fractures, mixed fractures were seen in two patients, one patient had longitudinal fracture, and one had transverse fracture. Sensorineural hearing loss was found in four ears, mixed hearing loss was found in one ear, conductive hearing loss was noticed in one ear, and two ears were found to be normal.

Sensorineural hearing loss was noticed in the patient who sustained a fracture of the occipital bone.

Out of the three patients who sustained parietal bone fractures two patients were found to have normal hearing, the third patient had sensorineural hearing loss in one ear which became normal and mixed hearing loss in the other ear that improved.

Bilateral sensorineural hearing loss was detected in patients who sustained facial bone fractures.

Only one patient with parietal bone fracture with normal hearing complained of vertigo in which positional test was negative and was treated conservatively.

Out of the four patients who fell from a height three patients had normal hearing one had conductive hearing loss. Parietal bone fracture was detected in one patient who had normal hearing. Hearing loss progressed in one patient.

Delayed facial nerve paresis was seen in one patient who presented with history of bull gore injury which recovered within 3 months.

Of the 60 patients 23 had loss of consciousness ranging from 5 minutes to 20 minutes, for 2 patients details were not available for loss of consciousness.

Of these 23 patients 20 had alleged history of RTA involving two wheelers, one had fall form height and one was involved in a four wheeler accident and one in a three wheeler accident.

Of these 60 patients 6 complained of hearing loss in both the ears and, 9 patients complained of vertigo. Only 2 patients complained of vertigo with hearing loss..

Dix-Hallpike positional test was positive in three patients for whom Epley's repositioning maneuver was done and rest of the patients were treated with labyrinthine suppressants.

One patient complained of tinnitus.

On examination hemotympanum was noticed in 2 patients which resolved over a period of three months. 1 patient presented with laryngeal trauma, rest of the patients had bleeding either from the nose or the ear.

Bleed from the ear was noticed to be due to laceration in the external auditory canal. Patients having ear bleed were called after a week for auditory assessment, no active intervention was needed. 1 patient had otitis externa which was treated conservatively.

DISCUSSION

This prospective study was done in a tertiary care teaching hospital to look at the incidence of hearing loss in patients who sustained minor head injury. The behavior of hearing loss was evaluated by serial assessment of hearing.

Road traffic accidents (RTA) were the most common cause of Minor head injury as seen in all studies. The incidence of road traffic accidents in age groups 20-50 years, in our study, was similar to the study done by Ludwig podoshin Error: Reference source not foundand M R Abd AL-HadyError: Reference source not found. Whereas in the study done by GriffithError: Reference source not found, the majority were seen in late teens.

Two wheeler accidents were found to be the commonest cause of RTA causing minor head injury in our study. where as George.G.BrowningError: Reference source not found reported that assault /fight was the major etiology causing minor head injury. 75% of the RTA's were two wheeler accidents and none of them were wearing helmets at the time of accident. In about 40% of our patient's consumption of alcohol would have contributed to the road traffic accident Error: Reference source not found Error: Reference source not found.

Symptoms of hearing loss were found in only 10% of patients which is in agreement with SchucknechtError: Reference source not found and Makashima Error: Reference source not founddue to the involvement in high frequency region.

But the symptoms of vertigo was found to be 15% which is low compared with TogliaError: Reference source not found (61%) and RosalynError: Reference source not found (95%).

Incidence of tinnitus also is low which is in agreement with GriffithError: Reference source not found, who reported no complaints of tinnitus.

The incidence of Hearing loss in our study is 62% which is in agreement with GriffithError: Reference source not found 56% and males being most affected.

The commonest type of hearing loss was sensorineural loss confined to high frequencies. Higher the frequency affected and severe the hearing loss the prognosis was poor. The degree of hearing loss determined the out come, and it was found in our study that patients who had moderate to severe hearing loss at the time of injury had a poorer prognosis.

Studies[77] [78]have shown that temporal bone fractures have a higher incidence of hearing loss, which was found to be the case in our series also.

DPOAEs are present[79] [80] across most frequencies at and above 1000Hz in 99 to 100% of ears with normal hearing and they are absent when sensorineural hearing loss exceeds 40-50dBError: Reference source not found Error: Reference source not found which was similar in our study. No studies were found in literature where DPOAE was assessed in minor head injury.

It was seen that DPOAE was absent in 38.6% at 1000Hz, 36% at 2000Hz, 29.8% at 4000Hz in patients even with normal PTA thresholds after mild head injury.

In case of Mild hearing loss on PTA, there was absence of emissions in 70% at 1000 Hz, 69% at 2000Hz, 83% at 4000Hz. This would suggest that damage to outer hair cells becomes more pronounced when there is manifest hearing loss on PTA. In few cases with normal hearing, DPOAEs were absent through out the evaluation time period suggesting irreversible damage to outer hair cells

As the hearing improved oto-acoustic emissions were detectable, however in cases where the hearing loss progressed, emissions could not be recorded. Changes in DPOAEs were found to be statistically significant only at 3000 Hz (p value-0.002) and 4000Hz (p value-0.003), in mild head injury.

SUMMARY

Hearing loss was detected in 62% of patients who sustained minor head injury, though only 10% of them complained of hearing loss.

The commonest cause of Head injury was Road traffic Accidents and the most involved were two wheeler riders with out helmets.

The degree of hearing loss and the frequency affected determined the prognosis in these patients.

This study showed that DPOAE assessment at 3000 Hz and 4000 Hz is significant and has a higher predictive value in assessing outer hair cell damage when compared to Pure Tone Audiometry.

LIMITATIONS

19 out of 60 were lost to follow-up. Of the remaining only 68% patients came for first follow up and 30% for the second evaluation.

CONCLUSION

Hearing should be evaluated in all patients with head injury even if they do not complain of hearing loss, as a seemingly minor hearing loss at a very young age becomes severe as age progresses, when combined with other factors which affect hearing.

DPOAE should be done in evaluating patients with minor head injury and test need to be done at 3000 Hz and 4000 Hz only, suggesting that time and resources may be saved by restricting the tests to those frequencies.

This study has shown that Head injury could have been avoided in all patients who had two wheeler accidents if they were wearing crash helmets.

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